Management of a patient with recurrent stent thrombosis: from diagnosis to treatment

To the editor We have recently read with great interest the article by Chyrchel et al[1] entitled "Recurrent stent thrombosis in a patient with neurofibromatosis type 1." We appreciate the authors for the successful treatment of recurrent stent thrombosis (ST) in a patient with neurofibromatosis type 1. On the other hand, we believe that there are several major drawbacks that need to be addressed.

First of all, ST is a rare but fatal complication following percutaneous coronary interventions (PCI).[2] Despite technological advances in PCI, this complication is still associated with significant morbidity and mortality. Although the pathophysiology of ST is versatile, most STs depend on the mechanical factors associated with the implanted stent (underexpansion, malapposition, edge-dissections, and residual inflow-outflow disease). Hence, intravascular imaging has been instrumental in optimizing the technique of coronary stenting as currently practiced.[3] The 2018 European Society of Cariology / European Association for Cardio-Thoracic Surgery guidelines on myocardial revascularization recommend intravascular imaging to prevent strut malapposition for PCI.[3] We know that suboptimal stent expansion is the single most important factor which has most strongly been associated with ST after PCI.[4] The readers may wonder whether mechanical factors of ST in a patient with recurrent ST were excluded by intravascular imaging methods (intravascular ultrasound or optical coherence tomography).

Second, the effect of hereditary coagulation disorders in the formation of ST has been previously evaluated in a limited number of studies. Specifically, G1691A Factor V Leiden, G20210A factor II mutation, and C677T homozygous methylenetetrahydrofolate reductase polymorphism were evaluated in patients with ST.[5] Although no statistically significant relationship was found between these gene variations and ST, there is a relatively high prevalence of at least 1 gene anomaly in the patient subset, suggesting that thrombophilia screening in ST cases may be justified.

Lastly, surgical revascularization may even be chosen initially to prevent failed PCI in such patients.

ARTICLE INFORMATION

AUTHOR NAMES AND AFFILIATIONS Ezgi G. Güner, Cemalettin Akman, Cemil Can, Ahmet Güner (Department of Cardiology, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey)

CORRESPONDENCE TO Ahmet Güner, MD, Department of Cardiology, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, 34303, Kucukcekmece, Istanbul, Turkey, phone: +90 5056533335, email: ahmetguner488@gmail.com

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Author's reply I have read with great attention the comment written by Güner et al to our article. The rate of stent thrombosis (ST) has been gradually reduced in the recent years by improving stent platforms, optimizing procedural techniques, and antiplatelet treatment. However, the problem has not been solved in its entirety. The rate of ST remains at the level

of 1% in patients with STEMI.[1] Due to the high rate of fatal consequences of ST (death, cardiogenic shock), every scientific discussion and proposal which could further reduce the ST rate is justified.

The main purpose of the article was to emphasize the potential role of vascular and histological pathology associated with rare neurofibromatosis disorders which could increase the risk of stent thrombosis independently of the well-known risk factors.[2] I could agree that advanced visualization techniques (intravascular ultrasound, optical coherence tomography) should be considered to exclude mechanical causes of stent thrombosis in patients with acute coronary syndrome.[3] On the other hand, technical faults of stent implantation: small diameter, stent underexpansion, incomplete stent struts apposition, or dissections are mostly responsible for early ST within first hours or days after the procedure.[4] In the presented case, the first ST occurred 3 years after the procedure and the second, 10 months after the procedure, which could point to another cause of ST.

Hereditary coagulation disorders and thrombotic factors insufficiency were investigated in relation to ST appearance in the recent years.[5] Coagulation disorders cannot be categorically excluded as a potential reason for stent thrombosis. In our case, they would probably reveal themselves earlier in youth or after the first implantation of stents 4 years before the presented events. In our institution, we screen patients towards hereditary coagulation disorders in case of atypical localization of thrombosis or acute coronary syndrome appearing before 40 years of age.

In my opinion urgent coronary artery bypass grafting in this clinical situation was not a reasonable therapeutic option. Acute myocardial infarction, aggressive antiplatelet and antithrombotic treatment that significantly increases risk of major hemorrhagic complications during a cardiac surgery have made this option debatable. Besides 1-vessel bypass grafting (except the left anterior descending artery) is very rarely performed in clinical practice even in stable patients.

ARTICLE INFORMATION

AUTHOR NAMES AND AFFILIATIONS Michał Chyrchel (2nd Department of Cardiology, Institute of Cardiology, Jagiellonian University Medical College, Kraków, Poland)

CORRESPONDENCE TO Michał Chyrchel, MD, PhD, 2nd Department of Cardiology, Institute of Cardiology, Jagiellonian University Medical College, ul. Jakubowskiego 2, 30-688 Kraków, Poland, phone: +48 12 400 22 51, email: mchyrchel@gmail.com

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